

Letter to the Editor

Does Aluminum Lactate Cause Spinal Cord Infarction in Rabbits?

Dear Editors:

Bombi and colleagues recently reported an interesting and important study of aluminum lactate and aluminum acetylacetonate as potential toxicants to rabbits (1). As stated by the authors, the role of aluminum in central nervous system diseases (Alzheimer's disease, dialysis dementia) is currently under intensive investigation. Therefore, my attention was drawn to their report of "posterior paraplegia with a wide infarcted area in the spinal cord" in two of three rabbits that had been injected intravenously with aluminum lactate in the form of a suspension of liposomes. It should be noted that no spinal cord infarct was found in the third rabbit, although it had received a much larger amount of inoculum, or in any of six rabbits that had been injected with much larger amounts of aluminum lactate as an aqueous solution. The authors hypothesize ischemia as an etiologic factor. They might have been thinking of embolic phenomena related to the injected suspension of lipid material. However, emboli might then have been expected in the brain even more than in the spinal cord, and especially in the third rabbit that received a much larger dose.

I would like to offer the alternative suggestion that the spinal cord infarcts may have been caused by accidental traumatic vertebral fracture or dislocation. It is known that sudden kicking or jumping movements by the rabbit's powerful hindlimbs can cause spinal fractures during forcible immobilization for injection procedures (2). Such occurrences can occur even with careful handling by experienced personnel, and they may not be recognized immediately (2: 445).

This alternative suggestion is offered to promote careful consideration before introducing these data as yet another facet in the complex issue of neurotoxicity from aluminum. In all other respects, the papers of Bombi and colleagues have presented valuable data on the toxicologic importance of the chemical speciation of aluminum (3,4), and we can confirm their concept in a completely different *in vivo* system involving skeletal muscle (5).

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REFERENCES

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Response

Dear Editors:

Seymour Levine's comments on our paper (1) are in principle correct. However, not only did we use all possible precautions for preventing accidental traumatic vertebral injuries in experimental animals, but we also carefully examined the condition of individual vertebrae of all investigated rabbits. Moreover, further toxicological data dealing with the spinal cord infarction will be the matter of a forthcoming publication.

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REFERENCE

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